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REVIEW

Hair cortisol, stress exposure, and mental health in humans: A systematic review

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Abstract The deleterious effects of chronic stress on health and its contribution to the development of mental illness attract broad attention worldwide. An important development in the last few years has been the employment of hair cortisol analysis with its unique possibility to assess the long-term systematic levels of cortisol retrospectively. This review makes a first attempt to systematically synthesize the body of published research on hair cortisol, chronic stress, and mental health. The results of hair cortisol studies are contrasted and integrated with literature on acutely circulating cortisol as measured in bodily fluids, thereby combining cortisol baseline concentration and cortisol reactivity in an attempt to understand the cortisol dynamics in the development and/or maintenance of mental illnesses. The studies on hair cortisol and chronic stress show increased hair cortisol levels in a wide range of contexts/situations (e.g. endurance athletes, shift work, unemployment, chronic pain, stress in neonates, major life events). With respect to mental illnesses, the results differed between diagnoses. In major depression, the hair cortisol concentrations appear to be increased, whereas for bipolar disorder, cortisol concentrations were only increased in patients with a late age-of-onset. In patients with anxiety (generalized anxiety disorder, panic disorder), hair cortisol levels were reported to be decreased. The same holds true for patients with posttraumatic stress disorder, in whom — after an initial increase in cortisol release — the cortisol output decreases below baseline.

The effect sizes are calculated when descriptive statistics are provided, to enable preliminary comparisons across the different laboratories. For exposure to chronic stressors, the effect sizes on hair cortisol levels were medium to large, whereas for psychopathology, the effect sizes were small to medium. This is a first implication that the dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis in the development and/or maintenance of psychopathology may be more

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subtle than it is in healthy but chronically stressed populations. Future research possibilities regarding the application of hair cortisol research in mental health and the need for multidisciplinary approaches are discussed.

Contents

1. Introduction

A frequently assessed hormone in psychoneuroendocrine research is cortisol. Cortisol is a glucocorticoid hormone and is released by the adrenal cortex through stimulation of the hypothalamic-pituitary-adrenal (HPA) axis. Cortisol is crucial for proper body and brain functioning as it regulates numerous basal processes such as fat and glucose metabolism, blood pressure, inflammatory and immune responses, and thereby aids the organism to flexibly adjust to environmental challenges (Marieb and Hoehn, 2007). It is also commonly known as *the* stress hormone because it is released in higher doses under stressful conditions. In the reaction to basically all stressors, two classes of hormones are released; catecholamines and glucocorticoids, and the speed and magnitudes of both parts depend on the specific stressor (Pacák and Palkovits, 2001). Catecholamines such as noradrenaline or adrenaline work via the nervous system and within seconds, thereby enabling immediate physical reactions associated with the flight-or-fight response (also known as acute stress reaction). Glucocorticoids such as cortisol act via the hormonal route and support the activity of catecholamines over the course of minutes or hours. Cortisol affects and enables the coordination of brain and body functions involved in coping with a stressor (de Kloet et al., 2005). Effective coping with a stressor involves the rapid activation of a stress response when it is needed, as well as the efficient termination afterwards. The increased secretion upon the appearance of a stressor can result in temporarily increased availability of energy by increased muscle strength, increased memory function, increased immunity, and decreased sensitivity to pain (Marieb and Hoehn, 2007). This increased release is under normal circumstances terminated by cortisol itself as its production is part of a negative feedback-loop involving all parts of the HPA axis. However, the initiation as well as the termination of the stress response is susceptible to dysregulation; these processes can be delayed, excessive, flattened, or prolonged (McEwen, 2003; Oitzl et al., 2010).

A stress response consists of three phases: stress reaction, recovery, and adaptation (Oitzl et al., 2010). The bodily processes that maintain homeostasis during the different stress phases are called "allostasis" (McEwen, 2003). A state of increased activity of one "mediator" such as cortisol is an "allostatic state". Accumulation of mediator dysregulations over time are called "allostatic load" and can result in receptor desensitization and tissue damage (McEwen, 2003). Allostatic load is for example reflected in a chronic dysregulation of the HPA axis. Functioning of the HPA axis (measured in cortisol concentrations) in psychiatric populations has frequently been subject to research, and both hypo- and hyperactivity of the HPA axis have been found in different psychiatric populations, for example in patients with depressive and anxiety disorders (Olff et al., 2006; Vreeburg et al., 2009, 2010), or with personality disorders (Lieb et al., 2004).

Until a few years ago, cortisol has solely been analyzed from blood serum, saliva, or urine. These analyses offer the possibility to explore the dynamics and the concentration of acutely (serum, saliva) or short-term (urine) circulating cortisol concentrations. Studies using these methods have provided insight into dysregulations of stress reactions and have established the connection between HPA axis activity alterations and mental illness. For example, dysregulation of the HPA axis is supposed to be an important causal factor in the development of panic disorder (de Kloet et al., 2005), which